Nutritional Status of Vitamin B12 and Vitamin D: Important Indicators of Cancer Risk

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A recent article by Drs. A.S. Plant and G. Tisman, published in Nutrition and Cancer, highlights the emerging evidence that links suboptimal nutritional status of vitamin B\textsubscript{12} and vitamin D with increased risk of cancer.\textsuperscript{1} Previous studies have shown that low serum levels of vitamin D (25-hydroxyvitamin D) are associated with a higher frequency of at least 17 different malignancies, including breast, colon, prostate, ovarian and other cancers.\textsuperscript{2-7} Vitamin B\textsubscript{12} deficiency has been associated with an increased risk of breast cancer.\textsuperscript{8,9} In one study of postmenopausal women, low levels of vitamin B\textsubscript{12} were associated with a 2.5 - 4.0 increased risk of breast cancer.\textsuperscript{10}

The anticancer properties of vitamin D are reported to stem from its ability to bind to vitamin D receptors on the surface of cells (many tissues contain vitamin D receptors) and thereby, decrease the rate of cellular proliferation (cellular replication) and encourage cellular differentiation (maturation). Both of these influences reduce the risk that a cell will become malignant. Vitamin D also has been shown to exert antineoplastic influences on existing cancer cells, which maintain vitamin D receptors. Under experimental conditions, vitamin D has been able to slow the growth of cancer cells and encourage cellular differentiation (more mature-looking cells with less malignant properties). Several recent clinical trials have shown that high-dose vitamin D supplementation can slow the rise in PSA in prostate cancer patients and improve median survival when added to conventional therapies. Vitamin D also binds to receptors on immune cells, acting as an immune modulator.\textsuperscript{11-15}

The anticancer properties of vitamin B\textsubscript{12} are considered to be related to its involvement in cycling homocysteine to methionine. Methionine is then converted into S-adenosyl methionine (SAMe), which provides a methyl group to form the methylated nucleotides required for DNA synthesis. As cells replace themselves from one generation to the next, sufficient vitamin B\textsubscript{12} and folic acid are required to enable the emerging cell to make sufficient and well-formed DNA. Even a marginal deficiency in vitamin B\textsubscript{12} and/or folic acid can lead to hypomethylation of DNA. DNA hypomethylation makes the chromosomal linkages weaker and more susceptible to forming mutations linked to cancer. Many cancers have been linked to DNA
hypomethylation of oncogenes suppressor genes, which has been shown to be prevalent in patients with low levels of vitamin B$_{12}$. Recent evidence suggests this appears to be especially critical in the prevention of breast cancer.\textsuperscript{8,9,10,16,17}

**Demand for Vitamin B$_{12}$ and Vitamin D Increases With Age**

The incidence of many cancers increases with advancing age. Although there are numerous reasons to explain this finding, one factor may be related to the fact that nutritional status of vitamin B$_{12}$ and vitamin D tends to be reduced as we age, due to predictable age-related changes in the body’s physiology. In the case of vitamin B$_{12}$, 30 percent to 40 percent of elderly people suffer from gastritis, an inability to secrete adequate stomach acid, which impairs absorption of vitamin B$_{12}$. Stomach acid secretions include intrinsic factor, which binds with vitamin B$_{12}$ to facilitate its absorption into the bloodstream upon reaching the ileum of the small intestine. As such, the age-related decrease in stomach acid secretion results in reduced absorption of vitamin B$_{12}$ as we age.\textsuperscript{18}

In regard to vitamin D, as we age, our skin shows a reduced ability to synthesize vitamin D upon exposure to sunlight (ultraviolet radiation). The kidneys display a reduced ability to convert 25-hydroxyvitamin D into 1,25-dihydroxyvitamin D (calcitriol), which is the most potent form of vitamin D. As a result, our vitamin D status tends to decrease with age.\textsuperscript{19} Studies demonstrate that the frequency of serum vitamin D insufficiency (25-hydroxyvitamin D) is highest among the elderly. As much as 50 percent of the elderly population in North America may have blood levels of 25-hydroxyvitamin D below 75 nmol/L. This not only increases risk of osteoporosis and related fractures, but also may increase risk of certain cancers.\textsuperscript{20}

**Findings From the Plant and Tisman Study of Cancer Patients**

In a study of 70 cancer patients, blood results showed that 72 percent of patients had blood levels of 25-hydroxyvitamin D below 75 nmol/L and 34 percent had blood levels of holotranscobalamin (HTCII) below 69 pg/ml. Holotranscobalamin is the metabolically active form of vitamin B$_{12}$.\textsuperscript{1} This is an important point, as only 6 percent of this patient group showed low levels of serum vitamin B$_{12}$ (normal = $>$300 pg/ml). Thus, serum levels of holotranscobalamin are considered a more accurate indicator of vitamin B$_{12}$ status and cancer risk. The results of this study indicate that many cancer patients have low blood levels of vitamin D and/or vitamin B$_{12}$ (HTCII), which may have made these individuals more susceptible to cancer
development.  

Previous studies have shown that individuals with low serum levels of vitamin D (below 75 nmol/L) and/or hypomethylation of DNA, secondary to compromised nutritional status of folic acid and/or vitamin B$_{12}$, are more susceptible to subsequent development of certain cancers. The findings of Plant and Tisman serve to support the correlation between suboptimal nutritional status of vitamin D and vitamin B$_{12}$, and increased cancer risk.

Butterworth, et al., found the cervical dysplasia risk from human papillomavirus (HPV) to be limited to those with low folic acid status. Reversal of cervical dysplasia and bronchial dysplasia (two precancerous states) has been attained through the administration of folic acid and vitamin B$_{12}$ supplementation.  

Based on the available evidence, it is advisable to include fasting vitamin D, holotranscobalamin and serum folic acid levels among the battery of blood tests performed to help determine a patient’s health status and risk of future illness. Many health experts suggest that vitamin D levels (25-hydroxyvitamin D) should be in the range of 80 nmol/L to 130 nmol/L, and transcobalamin levels should be in the range of 70 pg/ml to 130 pg/ml. Vitamin B$_{12}$ levels should exceed 300 pg/ml. To maintain these values throughout adult life, it may be wise for individuals to supplement each day with 400 IU to 1,000 IU of vitamin D (depending on their initial fasting levels) and a B-50 complex (containing 50 mcg of vitamin B$_{12}$ and 400 mcg of folic acid). Studies suggest that a folic-acid fasting blood level of less than 4 ng/mL is the level at which chromosomal breaks occur more frequently, upon which cancer risk increases dramatically. Currently, 15 percent of the U.S. population has folic acid levels below 4 ng/mL. Low intake levels and serum levels of folic acid appear to especially increase risk of colon cancer. Some studies suggest that an intake of folic acid above 400 mcg per day may reduce risk of colon cancer by 31 percent.

Dr. Tisman recommends supplementing cancer patients with 500 mcg to 1,000 mcg of vitamin B$_{12}$ (orally), especially if they are undergoing chemotherapy, and 1,000 IU to 4,000 IU of vitamin D (orally). Monitoring of blood levels is necessary in these cases to ensure that HCTII blood levels are maintained between 70 pg/ml and 130 pg/ml, and that 25-hydroxyvitamin D levels do not exceed 200 nmol/L. Previous studies have shown that co-administration of vitamin B$_{12}$ and folic acid with certain chemotherapy drugs (e.g., Alimta) reduced mucositis and diarrhea, which normally occur with these drugs. Vitamin B$_{12}$ and folic acid play an important role in supporting the integrity of the intestinal lining. Higher doses of these nutrients are required.
when the body is under attack from chemotherapy agents, which can damage these tissues.¹

References


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